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NEWS 1		Web Page URLs for STN Seminar Schedule - N. America
NEWS 2	Jan 25	BLAST(R) searching in REGISTRY available in STN on the Web
NEWS 3	Jan 29	FSTA has been reloaded and moves to weekly updates
NEWS 4	Feb 01	DKILIT now produced by FIZ Karlsruhe and has a new update frequency
NEWS 5	Feb 19	Access via Tymnet and SprintNet Eliminated Effective 3/31/02
NEWS 6	Mar 08	Gene Names now available in BIOSIS
NEWS 7	Mar 22	TOXLIT no longer available
NEWS 8	Mar 22	TRCTHERMO no longer available
NEWS 9	Mar 28	US Provisional Priorities searched with P in CA/CAplus and USPATFULL
NEWS 10	Mar 28	LIPINSKI/CALC added for property searching in REGISTRY
NEWS 11	Apr 02	PAPERCHEM no longer available on STN. Use PAPERCHEM2 instead.
NEWS 12	Apr 08	"Ask CAS" for self-help around the clock
NEWS 13	Apr 09	BEILSTEIN: Reload and Implementation of a New Subject Area
NEWS 14	Apr 09	ZDB will be removed from STN
NEWS 15	Apr 19	US Patent Applications available in IFICDB, IFIPAT, and IFIUDB
NEWS 16	Apr 22	Records from IP.com available in CAPLUS, HCAPLUS, and ZCAPLUS
NEWS 17	Apr 22	BIOSIS Gene Names now available in TOXCENTER
NEWS 18	Apr 22	Federal Research in Progress (FEDRIP) now available
NEWS 19	Jun 03	New e-mail delivery for search results now available
NEWS 20	Jun 10	MEDLINE Reload
NEWS 21	Jun 10	PCTFULL has been reloaded
NEWS 22	Jul 02	FOREGE no longer contains STANDARDS file segment

NEWS EXPRESS	February 1 CURRENT WINDOWS VERSION IS V6.0d, CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP), AND CURRENT DISCOVER FILE IS DATED 05 FEBRUARY 2002
NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS INTER	General Internet Information
NEWS LOGIN	Welcome Banner and News Items
NEWS PHONE	Direct Dial and Telecommunication Network Access to STN
NEWS WWW	CAS World Wide Web Site (general information)

Enter NEWS followed by the item number or name to see news on that specific topic.

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FILE 'HOME' ENTERED AT 14:47:16 ON 11 JUL 2002

=> FIL BIOSIS MEDLINE SCISEARCH CA
COST IN U.S. DOLLARS

	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'BIOSIS' ENTERED AT 14:47:29 ON 11 JUL 2002
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FILE 'MEDLINE' ENTERED AT 14:47:29 ON 11 JUL 2002

FILE 'SCISEARCH' ENTERED AT 14:47:29 ON 11 JUL 2002
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FILE 'CA' ENTERED AT 14:47:29 ON 11 JUL 2002
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=> s p53
L1 105920 P53

=> s l1 and anxi?
L2 4 L1 AND ANXI?

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3 4 DUP REM L2 (0 DUPLICATES REMOVED)

=> d l3 1-4 ibib abs

L3 ANSWER 1 OF 4 CA COPYRIGHT 2002 ACS
ACCESSION NUMBER: 132:136036 CA
TITLE: Animals deficient in **p53** showing memory deficiency or behavioral disorders and their use as disease models
INVENTOR(S): Amson, Robert; Lassalle, Jean-michel; Telerman, Adam
PATENT ASSIGNEE(S): Fondation Jean Dausset-Ceph, Fr.
SOURCE: PCT Int. Appl., 32 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: French
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000007438	A1	20000217	WO 1999-FR1828	19990726
W: CA, JP, US				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
EP 1102530	A1	20010530	EP 1999-932972	19990726
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: FR 1998-10076 A 19980805
WO 1999-FR1828 W 19990726

AB Mice homozygous or heterozygous for mutation in the **p53** show deficiencies in memory, learning, and other cognitive functions and so may be useful as disease models. Apoptosis was seen in the brains of the mice with the accumulation of .beta.-amyloid.

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 2 OF 4 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2001:80402 BIOSIS
DOCUMENT NUMBER: PREV200100080402
TITLE: Cellular mechanisms in the cyclic affective disorders.
AUTHOR(S): Post, R. M. (1); Leverich, G. S. (1); Weiss, S. R. B. (1); Speer, A. M. (1); Obrocea, G. (1); Denicoff, K. D. (1)
CORPORATE SOURCE: (1) Biological Psychiatry Branch, National Institute of Mental Health, NIH, 10 Center Drive, Bldg. 10, Room 35239, Bethesda, MD, 20892 USA
SOURCE: Acta Neurologica Scandinavica, (2000) Vol. 102, No. Supplementum 175, pp. 15-17. print.
ISSN: 0001-6314.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English

L3 ANSWER 3 OF 4 MEDLINE
ACCESSION NUMBER: 1998295607 MEDLINE
DOCUMENT NUMBER: 98295607 PubMed ID: 9633850
TITLE: Management of borderline tumors of the ovary: state of the art.
AUTHOR: Trope C; Kaern J
CORPORATE SOURCE: The Norwegian Radium Hospital, Montebello, Oslo.
SOURCE: SEMINARS IN ONCOLOGY, (1998 Jun) 25 (3) 372-80. Ref: 60
Journal code: 0420432. ISSN: 0093-7754.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199806
ENTRY DATE: Entered STN: 19980713
Last Updated on STN: 19980713
Entered Medline: 19980630

AB Evidence published during several decades has shown that there is a group of epithelial ovarian tumors having histological and biological features between those of clearly benign and frankly malignant tumors. In 1963, FIGO accepted an intermediate group of ovarian carcinomas of low malignant potential. In 1973, WHO adopted the term borderline malignancies to describe these tumors. Borderline tumors represent approximately 10% to 15% of all epithelial ovarian malignancies. There are considerable discrepancies in the reported incidence of ovarian tumors of borderline malignancies. Some centers do not recognize tumors of this type and include them among invasive cancers. The prognosis for patients with borderline tumors is generally considered to be excellent. Although the standard treatment for older patients is abdominal hysterectomy and bilateral salpingo-oophorectomy, many young patients who have not completed childbearing can be safely treated with unilateral salpingo-oophorectomy coupled with comprehensive surgical staging, thereby preserving fertility potential. Even ovarian cystectomy has been reported, but the recurrence rate in the ovary approximates 15%. Many experts strongly believe that surgery is the only effective treatment for borderline tumors. Others routinely use postoperative chemotherapy for at least some subsets of patients with peritoneal implants. Currently, insufficient information is available to make a definitive statement regarding the efficacy of postoperative therapy. Nevertheless, clinicians are faced with the difficult task of making treatment recommendations to **anxious** patients. In the past, extensive application of automated methods for analytical cytology has resulted in large quantities of data on ploidy abnormalities in different types of human cancers. The main purpose has been to obtain additional parameters for the characterization

of various types of malignancy to give more precise information on their biological behavior. Data from the Norwegian Radium Hospital showed that the majority of borderline tumors have DNA diploid tumors and good prognosis, DNA aneuploidy indicates high risk. Several other investigators have shown the same results on DNA ploidy as a predictor of recurrence and survival, but a few others have shown conflicting results. Early studies suggest that **p53** mutation does not appear to play a role in the pathogenesis of these tumors. Studies on other molecular markers have not yet uncovered a reliable predictor of biologic behavior. However, it is hoped that future studies of genetics and molecular biology of these tumors will lead to useful laboratory tests.

L3 ANSWER 4 OF 4 MEDLINE
ACCESSION NUMBER: 83240683 MEDLINE
DOCUMENT NUMBER: 83240683 PubMed ID: 6134788
TITLE: Irreversible binding of [3H]flunitrazepam to different proteins in various brain regions.
AUTHOR: Sieghart W; Drexler G
SOURCE: JOURNAL OF NEUROCHEMISTRY, (1983 Jul) 41 (1) 47-55. Journal code: 2985190R. ISSN: 0022-3042.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198308
ENTRY DATE: Entered STN: 19900319
Last Updated on STN: 19970203
Entered Medline: 19830811

AB Irreversible photolabeling by [3H]flunitrazepam of four proteins with apparent molecular weights 51,000 (P51), 53,000 (**P53**), 55,000 (P55), and 59,000 (P59) was investigated in various rat brain regions by SDS-polyacrylamide gel electrophoresis, fluorography, and quantitative determination of radioactivity bound to proteins. On maximal labeling of these proteins, only 15-25% of [3H]flunitrazepam reversibly bound to membranes becomes irreversibly attached to proteins. Results presented indicate that for every [3H]flunitrazepam molecule irreversibly bound to membranes, three molecules dissociate from reversible benzodiazepine binding sites. This seems to indicate that these proteins are either closely associated or identical with reversible benzodiazepine binding sites, and supports the hypothesis that four benzodiazepine binding sites are associated with one benzodiazepine receptor. When irreversible labeling profiles of proteins P51, **P53**, P55, and P59 were compared in different brain regions, it was found that labeling of individual proteins varied independently, supporting previous evidence that these proteins are associated with distinct benzodiazepine receptors.

=> d his

(FILE 'HOME' ENTERED AT 14:47:16 ON 11 JUL 2002)

FILE 'BIOSIS, MEDLINE, SCISEARCH, CA' ENTERED AT 14:47:29 ON 11 JUL 2002
L1 105920 S P53
L2 4 S L1 AND ANXI?
L3 4 DUP REM L2 (0 DUPLICATES REMOVED)

=> s l1 and (memor? or learn?)
L4 330 L1 AND (MEMOR? OR LEARN?)

=> s l4 and p53 (5a) (muta? or non#function? or knockout)
'#' TRUNCATION SYMBOL NOT VALID WITHIN 'NON#FUNCTION?'
'#' TRUNCATION SYMBOL NOT VALID WITHIN 'NON#FUNCTION?'
'#' TRUNCATION SYMBOL NOT VALID WITHIN 'NON#FUNCTION?'

```
'#' TRUNCATION SYMBOL NOT VALID WITHIN 'NON#FUNCTION?'
The truncation symbol # may be used only at the end of a search term.
To specify a variable character within a word use '!', e.g., 'wom!n'
to search for both 'woman' and 'women'.  Enter "HELP TRUNCATION" at an
arrow prompt (=>) for more information.

=> s l4 and p53 (5a) (muta? or non-function? or knockout)
L5          85 L4 AND P53 (5A) (MUTA? OR NON-FUNCTION? OR KNOCKOUT)

=> s l4 and p53 (3a) (muta? or non-function? or knockout)
L6          83 L4 AND P53 (3A) (MUTA? OR NON-FUNCTION? OR KNOCKOUT)

=> s (p53 (3a) (muta? or non-function? or knockout)) (s) (inhib? (3a) (learn? or
memor?))
   3 FILES SEARCHED...
L7           0 (P53 (3A) (MUTA? OR NON-FUNCTION? OR KNOCKOUT)) (S) (INHIB?
               (3A) (LEARN? OR MEMOR?))

=> s (p53 (3a) (muta? or non-function? or knockout)) (s) ((inhib? or decreas? (3a)
(learn? or memor?))
UNMATCHED LEFT PARENTHESIS 'S) ((INHIB?'
The number of right parentheses in a query must be equal to the
number of left parentheses.

=> s (p53 (3a) (muta? or non-function? or knockout)) and ((inhib? or decreas?) (5a)
(learn? or memor?))
   3 FILES SEARCHED...
L8           0 (P53 (3A) (MUTA? OR NON-FUNCTION? OR KNOCKOUT)) AND ((INHIB? OR
               DECREAS?) (5A) (LEARN? OR MEMOR?))

=> s (p53 (3a) (muta? or non-function? or knockout)) and (learn? or memor?))
UNMATCHED RIGHT PARENTHESIS 'MEMOR?))'
The number of right parentheses in a query must be equal to the
number of left parentheses.

=> s (p53 (3a) (muta? or non-function? or knockout)) and (learn? or memor?)
L9          83 (P53 (3A) (MUTA? OR NON-FUNCTION? OR KNOCKOUT)) AND (LEARN? OR
               MEMOR?)

=> s (p53 (3a) (muta? or non-function? or knockout)) (p) (learn? or memor?)
L10         67 (P53 (3A) (MUTA? OR NON-FUNCTION? OR KNOCKOUT)) (P) (LEARN? OR
               MEMOR?)

=> s (p53 (3a) (muta? or non-function? or knockout)) (s) (learn? or memor?)
L11         59 (P53 (3A) (MUTA? OR NON-FUNCTION? OR KNOCKOUT)) (S) (LEARN? OR
               MEMOR?)

=> s (p53 (3a) (muta? or non-function? or knockout)) (5a) (learn? or memor?)
L12          7 (P53 (3A) (MUTA? OR NON-FUNCTION? OR KNOCKOUT)) (5A) (LEARN? OR
               MEMOR?)

=> dup rem l12
PROCESSING COMPLETED FOR L12
L13           4 DUP REM L12 (3 DUPLICATES REMOVED)

=> d l13 1-4 ibib abs

L13  ANSWER 1 OF 4  CA  COPYRIGHT 2002 ACS
ACCESSION NUMBER:         132:136036  CA
TITLE:                    Animals deficient in p53 showing memory deficiency or
                          behavioral disorders and their use as disease models
INVENTOR(S):              Amson, Robert; Lassalle, Jean-michel; Telerman, Adam
PATENT ASSIGNEE(S):       Fondation Jean Dausset-Ceph, Fr.
```

SOURCE: PCT Int. Appl., 32 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: French
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000007438	A1	20000217	WO 1999-FR1828	19990726
W: CA, JP, US				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
EP 1102530	A1	20010530	EP 1999-932972	19990726
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: FR 1998-10076 A 19980805
WO 1999-FR1828 W 19990726

AB Mice homozygous or heterozygous for **mutation** in the **p53** show deficiencies in **memory**, **learning**, and other cognitive functions and so may be useful as disease models. Apoptosis was seen in the brains of the mice with the accumulation of .beta.-amyloid.

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 2 OF 4 CA COPYRIGHT 2002 ACS
ACCESSION NUMBER: 130:79804 CA
TITLE: Mutation of the Angelman ubiquitin ligase in mice causes increased cytoplasmic p53 and deficits of contextual learning and long-term potentiation
AUTHOR(S): Jiang, Yong-hui; Armstrong, Dawna; Albrecht, Urs; Atkins, Coleen M.; Noebels, Jeffrey L.; Eichele, Gregor; Sweatt, J. David; Beaudet, Arthur L.
CORPORATE SOURCE: Department Molecular & Human Genetics, Baylor College Medicine, Houston, TX, 77030, USA
SOURCE: Neuron (1998), 21(4), 799-811
CODEN: NERNET; ISSN: 0896-6273
PUBLISHER: Cell Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The E6-AP ubiquitin ligase (human/mouse gene UBE3A/Ube3a) promotes the degrdn. of p53 in assocn. with papilloma E6 protein, and maternal deficiency causes human Angelman syndrome (AS). Ube3a is imprinted with silencing of the paternal allele in hippocampus and cerebellum in mice. The authors found that the phenotype of mice with maternal deficiency (m-/p+) for Ube3a resembles human AS with motor dysfunction, inducible seizures, and a context-dependent learning deficit. Long-term potentiation (LTP) was severely impaired in m-/p+ mice despite normal base-line synaptic transmission and neuroanatomy, indicating that ubiquitination may play a role in mammalian LTP and that LTP may be abnormal in AS. The cytoplasmic abundance of p53 was increased in postmitotic neurons in m-/p+ mice and in AS, providing a potential biochem. basis for the phenotype through failure to ubiquitinate and degrade various effectors.

REFERENCE COUNT: 69 THERE ARE 69 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 3 OF 4 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 1
ACCESSION NUMBER: 1996:319422 BIOSIS
DOCUMENT NUMBER: PREV199699041778
TITLE: Lessons from the p53 mutant mouse.
AUTHOR(S): Jacks, Tyler
CORPORATE SOURCE: Howard Hughes Med. Inst., Cent. Cancer Res., Mass. Inst.

Technology, Cambridge, MA 02139 USA
SOURCE: Journal of Cancer Research and Clinical Oncology, (1996) Vol. 122, No. 6, pp. 319-327. ISSN: 0171-5216.
DOCUMENT TYPE: General Review
LANGUAGE: English
AB The use of the mouse as a model organism in cancer research has a long and productive history, from the earliest studies of chemical carcinogenesis to the recent advances in gene targeting. Many of the basic principles of tumorigenesis have been formed in whole or in part through the study of tumor development in the mouse. Over the past decade, the major experimental approach has been to generate cancer-prone strains, either through transgenic technologies or, more recently, gene targeting. Here, I will review the state of the field of gene targeting of tumor-suppressor genes and concentrate on the **p53 mutant** strains and the lessons **learned** from the **p53 mutant** mouse.

L13 ANSWER 4 OF 4 CA COPYRIGHT 2002 ACS
ACCESSION NUMBER: 124:113776 CA
TITLE: The role of p53 in cancer development
AUTHOR(S): Oliner, Jonathan D.
CORPORATE SOURCE: Johns Hopkins University, USA
SOURCE: Sci. Am. Sci. Med. (1994), 1(4), 16-25 CODEN: SASMFP; ISSN: 1068-6746
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review with 9 refs. Few genes discovered in any field of research have achieved a level of notoriety equal to that of the p53 tumor suppressor. The reason for this is simple: p53 is more frequently mutated in human cancers than any other known gene. By examg. the interactions of p53 with other cellular mols., we are **learning** how **p53** works and why **mutating** it is advantageous to tumor cells. The knowledge derived from this work may be applied to the design of novel approaches to the diagnosis and treatment of cancer.

```
=> s (p53 (3a) (muta? or non-function? or knockout)) and (decreas? or inhib?) (5a)
(learn? or memor?)
   2 FILES SEARCHED...
L14          0 (P53 (3A) (MUTA? OR NON-FUNCTION? OR KNOCKOUT)) AND (DECREAS?
               OR INHIB?) (5A) (LEARN? OR MEMOR?)

=> s (p53 (3a) (muta? or non-function? or knockout)) and (decreas? or inhib?) (s)
(learn? or memor?)
   3 FILES SEARCHED...
L15          8 (P53 (3A) (MUTA? OR NON-FUNCTION? OR KNOCKOUT)) AND (DECREAS?
               OR INHIB?) (S) (LEARN? OR MEMOR?)

=> rem dup l15
DUP IS NOT VALID HERE
The DELETE command is used to remove various items stored by the
system.

To delete a saved query, saved answer set, saved L-number list, SDI
request, batch request, mailing list, or user-defined cluster, format,
or search field, enter the name.  The name may include ?  for left,
right, or simultaneous left and right truncation.

Examples:

  DELETE BIO?/Q          - delete query names starting with BIO
  DELETE ?DRUG/A         - delete answer set names ending with DRUG
```

```
  DELETE ?ELEC?/L         - delete L-number lists containing ELEC
  DELETE ANTICOAG/S       - delete SDI request
  DELETE ENZYME/B         - delete batch request
  DELETE .MYCLUSTER       - delete user-defined cluster
  DELETE .MYFORMAT        - delete user-defined display format
  DELETE .MYFIELD         - delete user-defined search field
  DELETE NAMELIST MYLIST - delete mailing list
```

To delete an ordered document or an offline print, enter its number.

Examples:

```
  DELETE P123001C         - delete print request
  DELETE D134002C         - delete document order request
```

To delete an individual L-number or range of L-numbers, enter the L-number or L-number range. You may also enter DELETE LAST followed by a number, n, to delete the last n L-numbers. RENUMBER or NORENUMBER may also be explicitly specified to override the value of SET RENUMBER.

Examples:

```
  DELETE L21              - delete a single L-number
  DELETE L3-L6            - delete a range of L-numbers
  DELETE LAST 4           - delete the last 4 L-numbers
  DELETE L33-             - delete L33 and any higher L-number
  DELETE -L55             - delete L55 and any lower L-number
  DELETE L2-L6 RENUMBER - delete a range of L-numbers and
                          renumber remaining L-numbers
  DELETE RENUMBER         - renumber L-numbers after deletion of
                          intermediate L-numbers
```

Entire sets of saved items, SDI requests, batch requests, user-defined items, or E-numbers can be deleted.

Examples:

```
  DELETE SAVED/Q - delete all saved queries
  DELETE SAVED/A - delete all saved answer sets
  DELETE SAVED/L - delete all saved L-number lists
  DELETE SAVED   - delete all saved queries, answer sets,
                   and L-number lists
  DELETE SAVED/S - delete all SDI requests
  DELETE SAVED/B - delete all batch requests
  DELETE CLUSTER - delete all user-defined clusters
  DELETE FORMAT  - delete all user-defined display formats
  DELETE FIELD   - delete all user-defined search fields
  DELETE SELECT  - delete all E-numbers
  DELETE HISTORY - delete all L-numbers and restart the
                   session at L1
```

To delete an entire multifile SDI request, enter DELETE and the name of the request. To delete a component from the multifile SDI, enter DELETE and the name of the component.

```
=> dup rem l15
PROCESSING COMPLETED FOR L15
L16           3 DUP REM L15 (5 DUPLICATES REMOVED)

=> d l16 1-3 ibib abs
```

L16 ANSWER 1 OF 3 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC. DUPLICATE 1
ACCESSION NUMBER: 2000:272158 BIOSIS
DOCUMENT NUMBER: PREV200000272158
TITLE: Behavioral alterations associated with apoptosis and down-regulation of presenilin 1 in the brains of p53-deficient mice.
AUTHOR(S): Amson, Robert; Lassalle, Jean-Michel; Halley, Helene; Prieur, Sylvie; Lethrosne, Florence; Roperch, Jean-Pierre; Israeli, David; Gendron, Marie-Claude; Duyckaerts, Charles; Checler, Frederic; Dausset, Jean; Cohen, Daniel; Oren, Moshe; Telerman, Adam (1)
CORPORATE SOURCE: (1) Fondation Jean Dausset-Centre d'Etude du Polymorphisme Humain, 27 Rue Juliette Dodu, 75010, Paris France
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (May 9, 2000) Vol. 97, No. 10, pp. 5346-5350. print..
ISSN: 0027-8424.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Presenilin 1 (PS1) expression is repressed by the p53 tumor suppressor. As shown herein, wild-type PS1 is an effective antiapoptotic molecule capable of significantly **inhibiting** p53-dependent and p53-independent cell death. We analyzed, at the functional and molecular levels, the brains of **p53 knockout** mice. Surprisingly, we found that lack of p53 expression induces apoptotic brain lesions, accompanied by **learning** deficiency and behavioral alterations. p53-deficient mice show an unexpected overexpression of p21waf1 with subsequent down-regulation of PS1 in their brains. This process is progressive and age-dependent. These data indicate that the p53 pathway, besides affecting tumor suppression, may play a major role in regulating neurobehavioral function and cell survival in the brain.

L16 ANSWER 2 OF 3 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 1998357083 MEDLINE
DOCUMENT NUMBER: 98357083 PubMed ID: 9692054
TITLE: Expression of p21WAF1/CIP1 is unrelated to p53 tumour suppressor gene status in oral squamous cell carcinomas.
AUTHOR: Yook J I; Kim J
CORPORATE SOURCE: Department of Oral Pathology, College of Dentistry, Yonsei University, Seoul, Korea.
SOURCE: ORAL ONCOLOGY, (1998 May) 34 (3) 198-203.
Journal code: 9709118. ISSN: 1368-8375.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199808
ENTRY DATE: Entered STN: 19980820
Last Updated on STN: 19980820
Entered Medline: 19980813
AB The p53 tumour suppressor gene is frequently mutated in oral squamous cell carcinomas. However, the downstream mechanism of p53 during oral carcinogenesis is not fully understood. The cyclin-dependent kinase **inhibitor** p21WAF1/CIP1 (p21), which can be induced by wild-type p53, functions as a downstream mediator of the antiproliferative and apoptosis-inducing actions of wild-type p53. To **learn** more about the roles of the p53 gene and its downstream mechanism, we evaluated **p53** gene **mutation** and immunohistochemical expression of p53 and p21 in 20 cases of oral squamous cell carcinoma. **p53** gene **mutations** were observed in 7 cases (35%). Overexpression of p53 was found in 4 of 13 cases with wild-type p53, and in 6 of 7 cases with **p53 mutations**. p21 expression was detected in 15

of 20 cases (75%). The expression of p21 correlated neither with **mutated p53 mutation** nor with **p53** protein overexpression. p21 was expressed even in carcinomas in which molecular analysis revealed a nonsense mutation. In normal oral mucosa, p21 expression was limited in the differentiating spinous cell layer. However, dysplastic or hyperplastic epithelium adjacent to the tumour demonstrated the increased expression of p21 even in the proliferating basal cell layer. These molecular and immunohistochemical data did not show any correlation with various clinico-pathologic parameters. These results suggest that **p53** gene **mutations** and altered expression of p21 are commonly involved in oral carcinogenesis, but do not correlate with each other or with the clinico-pathologic parameters. They also suggest that p21 expression in oral squamous cell carcinomas may be induced by a p53-independent pathway.

L16 ANSWER 3 OF 3 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 3
ACCESSION NUMBER: 1997:314116 BIOSIS
DOCUMENT NUMBER: PREV199799604604
TITLE: Geldanamycin-stimulated destabilization of **mutated p53** is mediated by the proteasome in vivo.
AUTHOR(S): Whitesell, Luke (1); Sutphin, Patrick; An, Wen G.; Schulte, Theodor; Blagosklonny, Mikhail V.; Neckers, Len
CORPORATE SOURCE: (1) AHSC, Room 3336, 1501 N. Campbell Ave., Tucson, AZ 85721 USA
SOURCE: Oncogene, (1997) Vol. 14, No. 23, pp. 2809-2816. ISSN: 0950-9232.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Mutation of the tumor suppressor gene p53 is the most common genetic abnormality detected in human cancers. Wild type p53 is a short-lived protein with very low basal intracellular levels. Most mutated forms of the protein, however, display markedly increased intracellular levels as an essential feature of their positive transforming activity. In this report, we have used selective **inhibitors** of the 20S proteasome to demonstrate that processing of p53 by ubiquitination and proteasome-mediated degradation is impaired by commonly occurring mutations of the protein. We found that this impairment of p53 turnover can be reversed by treatment of tumor cells with the benzoquinone ansamycin, geldanamycin, leading to a marked reduction in intracellular p53 levels. Finally, using cells which over-express a **mutant p53** protein, we were able to demonstrate that restoration of proteasome-mediated degradation by geldanamycin is accompanied by p53 polyubiquitination. Although much remains to be **learned** about the mechanisms involved, our data demonstrate that selective de-stabilization of mutant transforming proteins such as p53 can be achieved pharmacologically with agents such as geldanamycin which modify the function of molecular chaperone proteins within tumor cells.

=> s amson, R?/au; lassalle, J?/au; telerman, A?/au
L17 92 AMSON, R?/AU

LASSALLE, IS NOT A RECOGNIZED COMMAND
COMMAND STACK INTERRUPTED. ENTER "DISPLAY HISTORY"
TO SEE WHICH COMMANDS WERE EXECUTED.

The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s amson, R?/au;s lassalle, J?/au;s telerman, A?/au
L18 92 AMSON, R?/AU

L19 206 LASSALLE, J?/AU

L20 106 TELERMAN, A?/AU

=> s l18 or l19 or l20 and p53
L21 293 L18 OR L19 OR L20 AND P53

=> s (l18 or l19 or l20) and p53
L22 44 (L18 OR L19 OR L20) AND P53

=> s l22 and (anx? or (learn? or mem?)
UNMATCHED LEFT PARENTHESIS 'AND (ANX?'
The number of right parentheses in a query must be equal to the number of left parentheses.

=> s l22 and (anx? or (learn? or mem?))
L23 10 L22 AND (ANX? OR (LEARN? OR MEM?))

=> dup rem l23
PROCESSING COMPLETED FOR L23
L24 6 DUP REM L23 (4 DUPLICATES REMOVED)

=> d l24 1-6 ibib abs

L24 ANSWER 1 OF 6 MEDLINE
ACCESSION NUMBER: 2002188771 MEDLINE
DOCUMENT NUMBER: 21902089 PubMed ID: 11904448
TITLE: Wild-type and mutated presenilins 2 trigger **p53** -dependent apoptosis and down-regulate presenilin 1 expression in HEK293 human cells and in murine neurons.
AUTHOR: Alves da Costa Cristine; Paitel Erwan; Mattson Marc P; **Amson Robert; Telerman Adam**; Ancolio Karine; Checler Frederic
CORPORATE SOURCE: Institut de Pharmacologie Moleculaire et Cellulaire of Centre National de la Recherche Scientifique, Unite Mixte de Recherche 6097, Valbonne 06560, France.
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (2002 Mar 19) 99 (6) 4043-8. Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200204
ENTRY DATE: Entered STN: 20020403
Last Updated on STN: 20020425
Entered Medline: 20020424

AB Presenilins 1 and 2 are two homologous proteins that, when mutated, account for most early onset Alzheimer's disease. Several lines of evidence suggest that, among various functions, presenilins could modulate cell apoptotic responses. Here we establish that the overexpression of presenilin 2 (PS2) and its mutated form Asn-141-Ile-PS2 alters the viability of human embryonic kidney (HEK)293 cells as established by combined trypan blue exclusion, sodium 3'-[1-(phenylamino-carbonyl)-3,4-tetrazolium]-bis(4-methoxy-6-nitro)benzene sulfonic acid hydrate assay, and propidium iodide incorporation FACS analyses. The two parent proteins increase the acetyl-DEVD-al-sensitive caspase-3-like activity in both HEK293 cells and Telencephalon specific murine neurons, modulate Bax and bcl-2 expressions, and enhance cytochrome C translocation into the cytosol. We show that overexpression of both wild-type and mutated PS2

increases **p53**-like immunoreactivity and transcriptional activity. We also establish that wild-type- and mutated PS2-induced caspase activation is reduced by **p53** antisense approach and by pifithrin-alpha, a chemical inhibitor of **p53**. Furthermore, mouse fibroblasts in which the PS2 gene has been knocked out exhibited strongly reduced **p53**-transcriptional activity. Finally, we establish that the overexpression of both wild-type and mutated PS2 is accompanied by a drastic reduction of endogenous presenilin 1 (PS1) expression. Interestingly, pifithrin-alpha diminished endogenous PS2 immunoreactivity, whereas the inhibitor increases PS1 expression. Altogether, our data demonstrate that wild-type and familial Alzheimer's disease-linked PS2 trigger apoptosis and down-regulate PS1 expression through **p53** -dependent mechanisms.

L24 ANSWER 2 OF 6 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2002029242 MEDLINE
DOCUMENT NUMBER: 21625104 PubMed ID: 11752454
TITLE: Siah-1 binds and regulates the function of Numb.
AUTHOR: Susini L; Passer B J; Amzallag-Elbaz N; Juven-Gershon T; Prieur S; Privat N; Tuynder M; Gendron M C; Israel A; **Amson R**; Oren M; **Telerman A**
CORPORATE SOURCE: Molecular Engines Laboratories, 20 Rue Bouvier, 75011 Paris, France.
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (2001 Dec 18) 98 (26) 15067-72. Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200201
ENTRY DATE: Entered STN: 20020124
Last Updated on STN: 20020125
Entered Medline: 20020122

AB The Drosophila Seven in absentia (Sina) gene product originally was described as a protein that controls cell fate decisions during eye development. Its mammalian homolog, Siah-1, recently was found to be involved in **p53**-dependent and -independent pathways of apoptosis and G(1) arrest. We report that Siah-1 interacts directly with and promotes the degradation of the cell fate regulator Numb. Siah-1-mediated Numb degradation leads to redistribution of endogenous cell-surface Notch to the cytoplasm and nucleus and to augmented Notch-regulated transcriptional activity. These data imply that through its ability to target Numb for degradation, Siah-1 can act as a key regulator of Numb-related activities, including Notch signaling.

L24 ANSWER 3 OF 6 CA COPYRIGHT 2002 ACS
ACCESSION NUMBER: 132:136036 CA
TITLE: Animals deficient in **p53** showing **memory** deficiency or behavioral disorders and their use as disease models
INVENTOR(S): **Amson, Robert; Lassalle, Jean-michel ; Telerman, Adam**
PATENT ASSIGNEE(S): Fondation Jean Dausset-Ceph, Fr.
SOURCE: PCT Int. Appl., 32 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: French
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

WO 2000007438 A1 20000217 WO 1999-FR1828 19990726
W: CA, JP, US
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
EP 1102530 A1 20010530 EP 1999-932972 19990726
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI
PRIORITY APPLN. INFO.: FR 1998-10076 A 19980805
WO 1999-FR1828 W 19990726

AB Mice homozygous or heterozygous for mutation in the **p53** show deficiencies in **memory, learning,** and other cognitive functions and so may be useful as disease models. Apoptosis was seen in the brains of the mice with the accumulation of .beta.-amyloid.

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 4 OF 6 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 2
ACCESSION NUMBER: 2000:272158 BIOSIS
DOCUMENT NUMBER: PREV200000272158
TITLE: Behavioral alterations associated with apoptosis and down-regulation of presenilin 1 in the brains of **p53**-deficient mice.
AUTHOR(S): **Amson, Robert; Lassalle, Jean-Michel;** Halley, Helene; Prieur, Sylvie; Lethrosne, Florence; Roperch, Jean-Pierre; Israeli, David; Gendron, Marie-Claude; Duyckaerts, Charles; Checler, Frederic; Dausset, Jean; Cohen, Daniel; Oren, Moshe; **Telerman, Adam (1)**
CORPORATE SOURCE: (1) Fondation Jean Dausset-Centre d'Etude du Polymorphisme Humain, 27 Rue Juliette Dodu, 75010, Paris France
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (May 9, 2000) Vol. 97, No. 10, pp. 5346-5350. print..
ISSN: 0027-8424.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Presenilin 1 (PS1) expression is repressed by the **p53** tumor suppressor. As shown herein, wild-type PS1 is an effective antiapoptotic molecule capable of significantly inhibiting **p53**-dependent and **p53**-independent cell death. We analyzed, at the functional and molecular levels, the brains of **p53** knockout mice. Surprisingly, we found that lack of **p53** expression induces apoptotic brain lesions, accompanied by **learning** deficiency and behavioral alterations. **p53**-deficient mice show an unexpected overexpression of p21waf1 with subsequent down-regulation of PS1 in their brains. This process is progressive and age-dependent. These data indicate that the **p53** pathway, besides affecting tumor suppression, may play a major role in regulating neurobehavioral function and cell survival in the brain.

L24 ANSWER 5 OF 6 MEDLINE
ACCESSION NUMBER: 1998324754 MEDLINE
DOCUMENT NUMBER: 98324754 PubMed ID: 9662377
TITLE: Inhibition of presenilin 1 expression is promoted by **p53** and p21WAF-1 and results in apoptosis and tumor suppression.
AUTHOR: Roperch J P; Alvaro V; Prieur S; Tuynder M; Nemani M; Lethrosne F; Piouffre L; Gendron M C; Israeli D; Dausset J; Oren M; **Amson R; Telerman A**
CORPORATE SOURCE: Fondation Jean Dausset-CEPH (Human Polymorphism Study Center), Paris, France.
SOURCE: NATURE MEDICINE, (1998 Jul) 4 (7) 835-8.

Journal code: 9502015. ISSN: 1078-8956.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199904
ENTRY DATE: Entered STN: 19990420
Last Updated on STN: 19990420
Entered Medline: 19990407

AB Previously, we cloned a cDNA fragment, TSIP 2 (tumor suppressor inhibited pathway clone 2), that detects by northern blot analysis of M1-LTR6 cells a 3-kb mRNA downregulated during **p53**-induced apoptosis. Cloning the full-length TSIP 2 cDNA showed that it corresponds to the presenilin 1 (PS1) gene, in which mutations have been reported in early-onset familial Alzheimer's disease. Here we demonstrate that PS1 is downregulated in a series of model systems for **p53**-dependent and **p53** -independent apoptosis and tumor suppression. To investigate the biological relevance of this downregulation, we stably transfected U937 cells with antisense PS1 cDNA. The downregulation of PS1 in these U937 transfectants results in reduced growth with an increased fraction of the cells in apoptosis. When injected into mice homozygous for severe combined immunodeficiency disease (scid/scid mice), these cells show a suppression of their malignant phenotype. Our results indicate that PS1, initially identified in a neurodegenerative disease, may also be involved in the regulation of cancer-related pathways.

L24 ANSWER 6 OF 6 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1996:282894 BIOSIS
DOCUMENT NUMBER: PREV199699005250
TITLE: Isolation of 10 differentially expressed cDNAs in **p53**-induced apoptosis: Activation of the vertebrate homologue of the Drosophila seven in absentia gene.
AUTHOR(S): **Amson, Robert B.**; Nemani, Mona; Roperch, Jean-Pierre; Israeli, David; Bougueleret, Lydie; Le Gall, Isabelle; Medhioub, Monia; Linares-Cruz, Gustavo; Lethrosne, Florence; Pasturaud, Patricia; Piouffre, Laurence; Prieur, Sylvie; Susini, Laurent; Alvaro, Veronique; Millasseau, Philippe; Guidicelli, Catherine; Bui, Hung; Massart, Catherine; Cazes, Lucien; Dufour, Fabienne; Bruzzoni-Giovanelli, Heriberto; Owadi, Houman; Hennion, Claude; Charpak, Georges; Dausset, Jean; Calvo, Fabien; Oren, Moshe; Cohen, Daniel; **Telerman, Adam** **(1)**
CORPORATE SOURCE: (1) Foundation Jean Dausset-Centre d'Etude du Polymorphisme Humain, 27 rue Juleitte Dodu, 75010 Paris France
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1996) Vol. 93, No. 9, pp. 3953-3957.
ISSN: 0027-8424.
DOCUMENT TYPE: Article
LANGUAGE: English

AB We report the isolation of 10 differentially expressed cDNAs in the process of apoptosis induced by the **p53** tumor suppressor. As a global analytical method, we performed a differential display of mRNA between mouse M1 myeloid leukemia cells and derived clone LTR6 cells, which contain a stably transfected temperature-sensitive mutant of **p53**. At 32 degree C wild-type **p53** function is activated in LTR6 cells, resulting in programmed cell death. Eight genes are activated (TSAP; tumor suppressor activated pathway), and two are inhibited (TSIP, tumor suppressor inhibited pathway) in their expression. None of the 10 sequences has hitherto been recognized as part of the **p53** signaling pathway. Three TSAPs are homologous to known genes. TSAP1 corresponds to phospholipase C beta-4. TSAP2 has a conserved domain

homologous to a multiple endocrine neoplasia I (ZFM1) candidate gene. TSAP3 is the mouse homologue of the Drosophila seven in absentia gene. These data provide novel molecules involved in the pathway of wild-type **p53** activation. They establish a functional link between a homologue of a conserved developmental Drosophila gene and signal transduction in tumor suppression leading to programmed cell death.

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DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-2.95	-2.95

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